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UNIT CHARACTER VARIATION IN RODENTS[1]

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Recent progress in the study of the inheritance of coat colors in several species of rodents has revealed a rather striking similarity in the variations which have arisen in distinct species of that order. This similarity is not only a matter of appearance, which is familiar to all students of mammals, but extends as well to the manner of inheritance, and most recently has been found to characterize the localization of the determinants or genes for similar variations in two species. Such identity of cause of the same variation in two or more species indicates that such variations are homologous, and that the species which give rise to them have a relationship of a somewhat different and more intimate kind than that implied in the theory of relationship by common descent.

Before detailing the conditions in the species of rodents which have been studied, some explanation of the evidence and reasoning which underlie the localization of genes is due to the general reader. It is probably recognized by all students of biology that heritable variations arising generally by mutation are transmitted to the offspring in accordance with certain definite rules, known familiarly as Mendel's laws of inheritance. The chief of these laws states that heritable characters are transmitted as discrete units which segregate in the formation of the germ cells. A second principle asserts that the segre-

[1] In this paper, which is to regarded as a cursory survey leading to a consideration of one or two special points rather than as an authoritative exposition of variation in rodents, I have not felt it essential to furnish a detailed bibliography. The necessary references may be found in Castle (1920) and Morgan (1919) as noted in the bibliography, and an excellent survey in Wright (1917), which also considers the physiological and chemical aspects of color variation and inheritance.

gation of the units is independent, resulting in a random distribution of the characters of the parents among their gametes, such that when two units are involved the chances are equal that they will go together or separately. The first principle probably applies to all inheritance. Evidence has been brought forward to show that the factors or genes which represent the visible characters are not discrete but variable, and capable of change by selection, but this evidence has now been found to indicate not variability but plurality of units. The second principle still applies to most cases of inheritance but has been modified by the finding that two or more characters may not always be distributed independently but when entering a cross together may tend to stay together, and when entering a cross separately may tend to remain separate (in different individuals) in inheritance. This peculiarity was first remarked by Bateson (1906) in the case of the inheritance of flower color and pollen shape in sweet peas. In his experiments purple flower (as opposed to red) and long pollen (as opposed to round) appeared to be associated or coupled in crosses so that a marked distortion was evident in the second generation ratio of 9:3:3:1 expected on the usual hypothesis of independent assortment, in favor of the classes (purple-long and red-round) representing the grandparental combinations of these characters. The opposite phenomenon was noted and named repulsion. Later, Morgan in 1910 found the same phenomena while studying inheritance of certain characters which had arisen by mutation in the vinegar fly (*Drosophila melanogaster*). He conceived these two exceptions to Mendel's principle of independent assortment as two aspects of a single phenomenon which he termed *Linkage* or associated inheritance.

The interpretation of these events has constituted one of the great advances of biological science. Sutton, in 1902, suggested that the marked parallelism between the discreteness and assortment of unit characters and the behavior of the chromosomes might be due to the residence in the chromosomes of the determinants or genes representing unit characters. Immediately after Bateson's announcement of coupling, Locke (1906) pointed out the similarity between this new mode of inheritance and the results which might be expected if the coupled characters were determined in one chromosome. The development of this hypothesis, its proof and very important extension and generalization, have been the work of the American biologist T. H. Morgan, and of research workers associated with him, assisted more recently by data gathered by geneticists and cytologists working on many

species of plants and animals. There has resulted from this work the elaboration of the chromosome theory of heredity, for the details of which, and the supporting evidence, the interested reader must be referred to the original works, especially as summarized in two publications of Morgan and his co-workers (1915 and 1919).

For our purpose it is sufficient to note in brief that the theory supposes that the differential representatives of heritable characters are located in the nuclear material of the egg and sperm cells, more precisely in those remarkably constant and individual organizations of chromatin known as chromosomes, which appear at the time of cell division and which probably retain their individuality even in the resting stages of the nucleus. Of the evidence it must be observed that the interpretation and proof of the theory rest entirely on the study of linkage, or associated inheritance. This phenomenon is observed in the tendency which characters exhibit of remaining through several generations in their original combinations, resulting in an alteration of the expected Mendelian ratios based on independent assortment. This tendency may be absolute, in which case linkage is said to be complete. More often it is partial, that is, characters originally associated may separate in a certain proportion of instances, or characters originally separate may become associated. This change in the relationships of genes is known as "crossing-over" and it provides a quantitative measure of the strength of the tendency toward association. In terms of the chromosome hypothesis it is interpreted as an interchange of parts and of the genes which the parts carry, between two members of a chromosome pair, so that two genes originally resident in one chromosome may come to lie in two chromosomes and may thence be distributed to separate gametes and exhibit their effects (unit characters) in different individuals. For any two characters the number of times crossing-over occurs is found to have a characteristic value and this value is stated as the percentage of times crossing-over occurs as evidenced by the frequency of individuals possessing the two characters in the new combination. One other important aspect of these measurable breaks in linkage is that from the linkage strength may be inferred the proportional distance apart of linked genes. From cytological evidence crossing-over is supposed to take place between homologous chromosomes in the hybrid at the time when these chromosomes are intimately twisted one about the other. Breaks resulting in a separation of characters are then supposed on mere physical grounds to be more frequent between genes located far apart in the chromosomes

than between those located near together. The bulk of the evidence indicates that the loci of genes are on the same straight line in any chromosome. Numerical strength of linkage may then be a measure of the exact localization of the genes in the germ plasm, and it is to a consideration of this point that our whole discussion has led. For if the genes for unit characters can be thus localized, a direct comparison of species in which similar variations occur can be made on this point alone, even though the species cannot be crossed.

The study of localization of the genes for unit character variations is attended by numerous limitations. It can only be prosecuted through the experimental breeding of large numbers of organisms, exhibiting numerous variations. It is dependent even under these conditions on the occurrence of linkage, which is by no means common. It is a corollary of the location of genes in chromosomes, that the numbers of groups of linked genes be equal to the number of chromosome pairs present. Where the number of chromosomes is large, and the number of unit variations known is small, the chances are few that any two characters will be found to be localized in one chromosome pair. Even under such limitations, linked genes have been studied in several insects (chiefly *Drosophila*) and plants, and most recently in mammals. The general results of these studies have been to confirm the chromosome theory and to increase our knowledge of the localization of genes.

Correlative evidence has come from a brilliant series of cytological investigations on the germ cells of several organisms. It has been established that in the cells of each species are to be found a definite number of chromosomes, characteristic for the species. This number in germ cells is half the number found in the somatic cells, due to the intervention of reducing cell divisions. The chromosomes themselves are in general arranged in pairs of homologues in the somatic cells and in the primordial germ cells, one member of each pair having come from each parent, and this duality again becomes evidenced in the passage of one member of each pair into the germ cells which form the next generation. The individual chromosomes are sometimes recognizable by peculiarities of shape, etc. More often their constancy is of numbers only. These cytological results have been made possible only by a high development of technique and can provide even when greatly extended only correlative evidence on the localization of genes. The geneticist or cytologist no more expects to behold the gene of which his literature is full than the chemist hopes to see the atom of which he speaks with unabated glibness. The gene remains useful as a concept and a notation, doubly so now that it includes an idea of spatial definition.

The above is a somewhat pretentious introduction to a discussion which adds so little to the matters mentioned in the opening paragraph, and yet I hope it has not been without interest to those engaged in the study of mammalian variation and evolution. The facts and theories discussed are to have an important place in general biology, and one may perhaps wish to hear of progress in a field which has tended at times to shut itself off from its fellow branches, by the dialect it has been forced to use.

Perhaps the best way of presenting the evidence on unit variation in color in the Rodentia is to describe the appearance and genetic behavior of each of the principal variations with a short list of the species in which it has been studied, and of the species in which a variation of similar appearance has been reported.[2] Where the inheritance of a variation has not been determined by experimental breeding this fact is noted by an asterisk. This list makes no claim to completeness except in the cases of variations which have been studied experimentally. The rest of the variations have been reported as occurring in the wild or are represented by specimens in the Museum of Comparative Zoology at Harvard University, the Museum of the Boston Society of Natural History, or the American Museum of Natural History of New York. I am indebted to Dr. Glover M. Allen of the Boston Society of Natural History for help in gathering this part of the material, and for helpful suggestions and criticism of this paper.

All of the variations listed appear to have arisen, probably by mutation, from the primitive coat color of all rodents, the dull protective grey pattern known as "agouti." This color, which is actually a mosaic, is due to the presence of three pigments, black, brown and yellow, distributed uniformly over the dorsal surface of the animal. Each dorsal hair is characterized in general by an area of black next to the skin in which brown granules are mingled and generally masked by the black, followed by a band of diffuse yellow. The apex of the hair is typically black. The belly is always of a lighter shade than the dorsum, due to a lesser concentration of black pigment and a wider area of pale dusky yellow in the hairs. The "agouti" coat is seen in a typical form in the familiar wild house mouse (*Mus musculus*), the common rat of this country (*Rattus norvegicus*), etc. It characterizes the wild type forms of all the species included in the following list.

[2] This proceeding may be expected to lead to some errors since similarity of appearance is not always evidence of similarity in germinal constitution, but in the absence of breeding data we must use the only criterion available.

ALBINO

From this wild type distinct graded losses of pigment have taken place, the extreme of which is complete albinism, or entire absence of pigment, leaving the fur clear white and the eyes pink. The pinkness of the eye is due to the absence of pigment in the iris, which is typically colored by black or brown pigment granules, so that the blood in the capillaries on the retina is directly visible. This variation is to be sharply distinguished from "partial albinism," a term which has been applied, unwisely it now appears, to the occurrence of white spotting in animals whose eyes retain their full color. The color of the eyes is an important point of distinction between complete albinos and spotted animals. Cases of true albinism have been reported in nearly all the families of rodents. Data from only five of the commonest families are given here, the families being listed roughly in the order of their relationship from the more primitive to the more specialized.[3]

Leporidæ—	Oryctolagus cuniculus—European "rabbit."
Sciuridæ—	*Marmota monax—Woodchuck.
	*Sciurus hudsonicus—Northern red squirrel.[4]
	*Sciurus carolinensis leucotis—American gray squirrel.
	*Tamias striatus lysteri—Chipmunk.
Muridæ—	Mus musculus—House mouse.
	Rattus norvegicus—Common rat.
	*Microtus pennsylvanicus—Meadow vole.
	*Fiber zibethicus—Muskrat.
	Peromyscus leucopus noveboracensis—Deer mouse.
Hystricidæ—	*Erethizon dorsatum—Canada porcupine.
Caviidæ—	Cavia cobaya—Guinea-pig.

The inheritance of the albinism has been studied in the rabbit, the house mouse, the house rat, the deer mouse and the guinea-pig. In all of these it is due to a gene which acts as a Mendelian recessive to full color. At the same (albino) locus in the germ plasm have occurred other mutations. In the rat, a change in this locus has produced both

[3] I have followed the older order of classification which includes the Leporidæ in the Rodentia.

[4] Through the kindness of Professor Barrows of the Michigan Agricultural College and Prof. W. E. Castle of Harvard University the writer has learned of the capture of a pair of albino red squirrels by A. E. Secord, of Wheeler, Michigan. Breeding experiments to test the inheritance of this variation were to have been attempted but expense and pressure of other work have prevented the writer from undertaking the project. At last reports the squirrels were alive and for sale and it is hoped that they will come into possession of some interested person.

albino and its dominant allelomorph ruby-eyed dilute, in which the reduction of the melanic pigments is visible in the generally lighter tone of black, coupled with a complete absence of yellow. In the guinea-pig three graded variations have occurred: (1) *dilution*, resulting in a reduction of all pigments; (2) *ruby*, resulting in the absence of yellow, and the further reduction of black and brown in fur and eyes to very light shades (probably homologous with the ruby variation in rats); and (3) *Himalayan albinism*, which determines the absence of yellow and the restriction of black and brown to the extremities, ears, nose, feet, and rump, while the eyes are pink. These three conditions are distinct in appearance, do not blend in crosses and are all alternative allelomorphs with full color and with each other. No complete albinism is known in the guinea-pig. In the rabbit two changes have taken place: Himalayan albinism (probably homologous with the Himalayan albinism of guinea-pigs) and albinism. These are allelomorphic with full color and with each other; that is, crosses of full colored animals with albinos produce only full colored young and in the second generation only colored and albinos. The same is true of the cross colored × Himalayan, while the cross Himalayan × albino produces only Himalayan and in the second generation only Himalayan and albino. The occurrence of this variation in several species, its similarities in appearance and in inheritance, and finally the production at the same locus as indicated by allelomorphism of other similarly appearing variations indicate that the particular locus in the chromatin at which these mutations have occurred is common to a number of widely different species and although such a statement cannot be proved except by a study of linkage relations between this and other common loci, it seems very probable that albinism is homologous variation throughout the rodents and in the species studied is due to homologous genes.[5]

PINK-EYE

This name has been applied by geneticists to a unit character in rodents which is not a form of albinism, as the pinkness of the eye might indicate, but a distinct eye and fur character. Animals ex-

[5] Since this paper was written, a fourth allelomorph in the albino series in rabbits has been reported by Castle (Science, vol. 53, April 22, 1921, p. 387). This variation, now studied genetically for the first time, is known as "chinchilla" and differs from the wild gray or "agouti" coat color in the absence of yellow, and its replacement by white, and in the reduction of black to a slate blue.

hibiting this variation show a general quantitative reduction in the black and brown pigments in both fur and eyes. A certain amount of pigment is present in the iris but not enough to obscure the blood color of the retina. Yellow pigment is not affected. Pink-eyed animals with the "agouti" coat pattern therefore appear yellow since the black bases of the dorsal hairs are a reduced slaty or bluish tint and are covered by the fully intense yellow parts of the hair. Black animals with this variation are slaty or bluish all over in mice and a dirty near-white in rats and guinea-pigs. Its distinctness from albinism becomes evident when pink-eyed colored animals are crossed with albinos. The first generation offspring in this case are all as fully colored as the wild type and if inbred produce full colored, pink-eyed colored, and albino young.

The variation occurs in the following species:

Sciuridæ—	*Marmota monax.
Muridæ—	Mus musculus.
	Rattus norvegicus.
	*Microtus pennsylvanicus.
	*Fiber zibethicus.
Caviidæ—	Cavia cobaya.

Its occurrence in the species marked * is probable but is based only on museum specimens with the coat colors peculiar to pink-eyed animals. The eyes in the mounted specimens may or may not agree with the original.

Data on the localization of this variation are available in large numbers for mice, and in lesser amount for rats and guinea-pigs. In these species it is a simple Mendelian recessive to full color (dark-eye). In rats and mice it is certainly a homologous variation, in appearance, in inheritance and in localization, for a large amount of linkage data indicates that the genes for pink-eye and for albinism are located in the same chromosome and at about the same relative distance apart. This localizes both of these genes in both species, and leads to some interesting conclusions and speculations which will be more fully considered later. In guinea-pigs there is incomplete evidence concerning the location of the gene for pink-eye but some data which Dr. Sewall Wright has kindly extracted from his breeding records and sent to me indicate that the locus of pink-eye is not in the albino chromosome but elsewhere. As we shall see, this may prove just as instructive concerning the homologies between species in germinal constitution as the more definite localization of the gene in rats and mice.

YELLOW

The self or solid yellow coat coloration in rodents appears to be divisible as to its cause into two different categories. In the first of these may be placed those yellow varieties which have arisen by a change in a gene governing the *extension* of black and brown to the fur, and the alternative (allelomorphic) condition of *restriction* of these melanic pigments to the eye, while the pelt is yellow. In the presence of this gene (restriction [r]) the melanic pigments are probably not produced in sufficient amount or to a sufficient intensity to invade the fur, leaving the residual yellow which is present in all "agouti" animals in possession of the whole extent of the hair. This gene is recessive to full extension and is distinct in its inheritance from the gene which determines the barring of each hair in the agouti pattern. Animals may possess the gene for yellow, with the gene for "agouti" or without it. "Agouti" yellows have much lighter bellies than non-agouti yellows. Restricted yellow occurs in the following rodents:

Leporidæ—	Oryctolagus cuniculus.
Muridæ—	*Microtus pennsylvanicus.
	Peromyscus maniculatus gambeli.
	Rattus rattus.
	*Rattus alexandrinus × R. rattus.
Caviidæ—	Cavia cobaya.

The distinction between this yellow and the second type, about to be described, is made on grounds of the mode of inheritance only, for the appearance of the latter type is identical with that of the former. The pelt of the second type is yellow and the eyes are dark. The gene which differentiates it is however dominant over its allelomorphs agouti and non-agouti. This dominant yellow is known only in the house mouse and because of its peculiar mode of inheritance has been the subject of considerable research through a combination of genetic and embryological methods to which Castle, Little, Kirkham and others have contributed. Such investigations have established the following facts:

(1) Yellow house mice do not breed true but when bred together always produce yellow and non-yellow young in the ratio of 2:1.

(2) Litters from yellow by yellow are on the average 25 per cent smaller than litters from non-yellow varieties.

(3) In the uteri of yellow females pregnant by yellow males there have been found disintegrating embryos approaching 25 per cent of the total embryos.

Ordinary heterozygotes (hybrids in one character) when bred together produce 25 per cent pure dominants, 50 per cent heterozygotes, and 25 per cent pure recessives. In the offspring of yellow mice the two latter classes have appeared in the expected proportions; pure dominant yellows have never been disclosed by breeding tests. It has therefore been concluded that the 25 per cent missing from the litters, the 25 per cent of disintegrating embryos, and the 25 per cent of expected pure yellows are the same. The intrauterine death of this class has been supposed to be due to a recessive lethal gene which when received from both parents causes the death of the resulting zygote or individual. In every case this lethal gene has been transmitted with the gene for yellow. It may be either completely linked or identical with the gene for yellow. At any rate it is present at the same locus with the gene which determines yellow, and any individual which receives yellow from both parents receives likewise the lethal gene from both parents and is doomed to death before birth. As to why this combination of two lethal genes is fatal we are still in the dark.

This yellow gene and the lethal associated with it are known only in house mice,[6] and the restricted yellow of the other species has not been reported in house mice. They are probably not homologous variations in spite of their similarity in appearance. The "yellow" varieties of rats are not really to be classified with other yellow rodents since they are actually "agoutis", differentiated from the wild gray by the pink-eye gene (to which we have referred) or by the very similar red-eye gene which act selectively on the melanic pigments to reduce rather than restrict them.

WHITE-SPOTTING

Almost as common as albinism among rodents is the spotting of certain portions of the coat with white. The white areas are as devoid of pigment as in albinos but here the likeness ends. Genetically white-spotting and albinism are distinct and contrary to the popular belief are not quantitatively but qualitatively unlike. Albinism is fundamentally the loss or change of a factor for the development of a peroxidase essential to the production of any pigment (cf. Wright '17) and its effects are of a general nature throughout the pelt and eyes. Spotting appears to be a change in a factor governing the distribution

[6] Several other factors may modify the appearance of yellow in mice; for instance, certain darkening factors in the presence of the yellow gene produce the black-and-tan and sable varieties of mice, while intensifying factors in the presence of yellow produce the brighter orange or red varieties.

of the pigments in the pelage. When pigmentation is present all over the pelt the condition is known as self or not-spotted. Spotting is inherited independently of albinism, since certain albinos crossed with spotted animals throw only selfs, while other albinos derived from white-spotted colored stocks have given spotted offspring. An albino may therefore be genetically either self or spotted although unable to give evidence of this condition except in its offspring by a colored animal which supplies the gene for the development of color.

On the grounds of its inheritance white-spotting in rodents may be classified in three categories. The first of these is piebald or Dutch spotting, apparently due to a gene recessive to self coloration and probably independent of other coat color unit characters. It may thence be present with albinism, yellow, pink-eye, agouti or black (see below). Piebald animals may be characterized by a typical localization of the spotting in a belt or collar as in belted mice or Dutch rabbits; the spotting may be confined to the face ("white-face" mice), or it may be distributed in a fairly uniform dorsal pattern as in hooded rats.[7] On all of these types the white-spotting varies only within general limits. In other piebald mice and in guinea pigs especially it appears purely at random, in irregular blotches hardly approximating any pattern at all. The spotting may also vary in amount from a few white hairs to over half the surface of the animal, although in general the pigmented areas exceed the white portions in total size. The belly is likewise more susceptible to spotting than the dorsum. This variation has been noted many times in wild species and I am certain the present list which has been hastily compiled does not represent the true distribution of this variation among rodents in general.

Leporidæ—	Oryctolagus cuniculus.
Sciuridæ—	*Sciurus finlaysoni.
Muridæ—	Mus musculus.
	Rattus norvegicus.
	*Evotomys gapperi.
Caviidæ—	Cavia cobaya.

In the rabbit, rat, mouse, and guinea-pig the similarity of its inheritance points toward a homology in this variation. Data on its localization are lacking except that in mice it is probably not located in the same chromosome with albinism and pink-eye, nor with the black-eyed white-spotting about to be discussed. In rats and guinea-pigs

[7] A hooded *Microtus* has been noticed in the Museum of Comparative Zoology at Harvard University.

it is likewise not linked as far as is known with any other color variation, while in rabbits it may be a property of the same locus at which English or dominant spotting is determined.

The two other categories of spotting are peculiar each to a single species. The English broken spotting of domesticated rabbits is a Mendelian dominant to self-color and has no probable homologue in other species, while the black-eyed white-spotting of mice, likewise a dominant, is apparently peculiar to mice although wild rodents resembling this type have been reported (e.g., *Sciurus finlaysoni*). This last variety is interesting in that it is less pigmented than any other type of spotting studied, some black-eyed white-spotted mice having pigment only in the eyes, while the rest of the pelt is pure white. It is discontinuous with piebald spotting, and is, like yellow, an unfixable hybrid, always throwing, when bred pure, a ratio of two black-eyed whites to one piebald. The cause of this peculiarity has lately been traced to its association with another lethal factor which determines the death in utero of all pure black-eyed white zygotes.

BLACK

One other color variation is common enough in rodents to make comparison profitable. This is the discontinuous change from the "agouti" coat to one which is black all over and it is probably due in all the species in which it occurs to a gene determining the exclusive development of black and brown pigments. It is always present in wild "agouti" type rodents and its appearance alone is conditioned by the change producing non-agouti, or the absence of the "agouti" pattern. Its recessive allelomorph is brown, which has occurred in rabbits, mice, guinea-pigs and possibly in rats. This gene is probably not linked with any of the other known genes in mice, but its relationships in other species have not been studied. The variation from agouti to black occurs in the following species:

Leporidæ— Oryctolagus cuniculus.
*Lepus americanus virginianus—Eastern varying hare.
Sciuridæ— *Sciurus hudsonicus?
*Sciurus niger ludovicianus.
*Sciurus niger niger.
*Sciurus carolinensis leucotis.
*Tamias striatus lysteri.
Muridæ— Mus musculus.
Rattus norvegicus.
*Fiber zibethicus.
Caviidæ— Cavia cobaya.

Specimens of individuals of other species which are much darker than the wild type are often seen in museums, usually labelled "melano" or "melanic variation." Many of these should not, I believe, be assumed to represent the true black variation. Some, as in "melanic" squirrels of various species, prove on close examination to be only very much darkened "agoutis" in which the black portions of each hair have been extended at the expense of the yellow portions. This variation has been studied by Punnett in the rabbit and found to be due to a gene distinct from black which is allelomorphic with the extension-restriction pair of allelomorphs. Much darkened "agoutis" have been produced in mice by crossing intense blacks or black-and-tans (a darkened form of yellow) with wild agouti-colored mice. The darkness is due in this case to a series of modifying genes distinct from either black, "agouti" or yellow which in the presence of these genes bring about an increase in the amount and intensity of the black pigment granules and a reduction in yellow. Such extended or darkened "agoutis" can usually (though not always) be distinguished from black by the lighter belly which is typical of the "agouti" pattern.

In addition to the coat color and pattern variations discussed above, many others have occurred in rodents which have been bred in the laboratory, though they are as yet known in too few species to make comparisons profitable. One of these, dilution, is a unit character in mice and rabbits. In this variation, the pigment granules are clumped and reduced in distribution, producing when acting on black varieties the familiar maltese color of blue rabbits and mice. The maltese cat is the result of a similar variation from black. Dilution is a simple Mendelian recessive to full color. The red and black blotching of guinea-pigs, the ticking or banding of the belly hairs in guinea-pigs, and the white bellies of a fancy variety of "agouti" mice are also known to be Mendelian unit characters.[8]

SUMMARY

A summary of the preceding discussion shows that we have examined five of the commonest variations in rodents: albinism, pink-eye, yellow, white-spotting, and black. In all of these the inheritance is known for at least three species, and in general the variant is recessive to the wild type. Dominant variations have occurred often enough (such as

[8] For a fuller discussion of these the interested reader is referred to Chapter XII in Castle's "Genetics and Eugenics," 1920, and to the series of papers by Wright cited above.

"English spotting" in rabbits, and black-eyed white-spotting and yellow in mice) to make us chary about drawing any general conclusions concerning the occurrence of evolution purely by loss mutations from type. The generality that does appear is rather the widespread occurrence in this order of similar variations both under domestication when the animals are saved and bred, and in the wild, when usually only the stuffed specimens are preserved. There is implied in this similarity, which in certain cases amounts to a homology, a similarity in that part of the organism which is responsible for the variations, i.e. the germ plasm. It may be that we know in these days as little concerning the *causes* of variation as did naturalists in the days of Lamarck or of Darwin. We do at least know where the causes are to be sought, and, once having arisen by a mysterious occurrence called mutation, we have learned something of the manner in which the variations are inherited, and by a process of inference have been able to localize still more exactly the region of change. The only permissible generality, then, concerns a general similarity in the germ plasm and probably in the individual chromosomes of these many species of rodents. But in one case the similarity between species has been found to be more than general. It has been found to be quite a specific similarity.

If we examine this case in detail we find that in two distinct, intersterile species, mice and rats, two similarly appearing variations have occurred, albinism and pink-eye. In rats the genes for these variations are linked with a strength of about 21 per cent, which is possibly slightly in excess of the actual. In mice the linkage between these genes in something less than 15 per cent, which is based on observations of 6700 animals raised solely for the purpose of determining this linkage and is probably reasonably accurate. In terms of the chromosome hypothesis these facts mean that these two genes are present in the same chromosome in rats and mice, in rats at a distance of 21 units apart, in mice at a distance of about 15 units apart. The difference in location is so small that for practical purposes we can say that they are located at homologous points in the two species.

In guinea-pigs where both of these variations occur, there is incomplete evidence, but the data which Doctor Wright has supplied indicate that pink-eye and albinism in guinea-pigs are probably not linked and may therefore be determined in different chromosomes. This does not prove, however, that these variations in guinea-pigs are *not* the same as those in mice and rats. It may mean that the chromosome which contains both genes in the more primitive Muridæ may in the

more specialized Caviidæ be represented by two chromosomes, the sum of which rather than either one separately may be homologous with the one chromosome of mice and rats. Although this will be recognized as speculation, there is some slight evidence that in the evolution of the rodents a fractionation of chromosomes may have occurred, for the mice and rats have 19 (haploid) while the guinea-pigs have 28. In the rabbit (*Oryctolagus cuniculus*) a member now judged too primitive for the true rodents and recently placed in the Lagomorpha with the others of the old rodent suborder Duplicidentata, the chromosome number is probably 12. If this progressive increase in the number of chromosomes through the order Rodentia is found to be a fact and not a chance phenomenon associated with the smallness of the sample of four species from which our cytological evidence is drawn, it may furnish a very important clue to a series of evolutionary relationships of more than ordinary interest.

In the concluding chapter of his recent book Professor Morgan (1919) has referred to the possible evolutionary significance of the localization of genes as determined by the study of linkage. He has there reviewed some of the work on similar variations in several species of insects by Metz and Sturtevant, pointing out the difficulties to be encountered in applying this method to the analysis of species, chief of which is the necessity of establishing the same linear order in each species of the genes for similar variations. A species in his point of view, and in this he follows De Vries, may ultimately prove to be a "community of genes." We may expect evidence of this community in the variations which arise from time to time within the species, whether they be at the time of specific value or not. Such community is not to be inferred from mere similarity in appearance but must rest on a more real homology of germinal cause. This kind of similarity is now apparent between *Mus musculus* and *Rattus norvegicus*, which have varied so far from a common type that they are now inter-sterile and have been placed recently in different genera. Yet they have retained a genetic constitution so similar that it contains genes common to both species. Whether this is due to a community of descent in the terms of current evolutionary theory or to relationship through some other cause is one of the questions which genetics, aided by the chromosome notation, may be expected at some time to answer.

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A HYBRID DEER OF THE F_2 GENERATION

BY HARTLEY H. T. JACKSON

[*Plate 8*]

INTRODUCTION

Among deer hunters who search for their spoils on the eastern slopes of the Cascade Mountains in the state of Washington, it is quite generally known that in a certain region the mule deer (*Odocoileus hemionus hemionus*) and the Columbian black-tailed deer (*Odocoileus columbianus columbianus*) hybridize. This area is where the western range of of the mule deer and the eastern range of the black-tail overlap. It may be roughly outlined by the summit of the Cascade Mountains on the west; the region of Stampede (or Yakima) Pass and lakes Keeches and Keechelus on the north; a line drawn north and south through a point 6 miles east of Signal Peak, the Tieton Basin, and Frost Mountain on the east; and Mount Adams on the south. Mr. James Henderson writes:

> This cross breeding of the mule deer and the Columbia black-tailed deer is not common. . . . . The different varieties of deer have their respective ranges very well defined, the mule deer seldom going to the summit of the divide and never, to my knowledge, beyond on the west slope. They are very much more scarce than the black-tails near the summit. I believe the lack of mates of their own kind leads the bucks of this variety to cross with the does of the black-tailed kind. Their offspring will then mate with either. (Letter to the U. S. Biological Survey from James Henderson, Mabton, Washington, April 24, 1917.)